WEST Search History

DATE: Thursday, January 30, 2003

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=OR		
L22	L21 not l18	3	L22
L21	L20 and switching	3	L21
L20	L19 and cd1$4	33	L20
L19	424/142.1 ((156.1/)!.CCLS.) and (l3 and l7)	221	L19
L18	L14 and (sle or lupus)	6	L18
L17	L16 and sle	1	L17
L16	5679347.pn.	2	L16
L15	l2 and cd1$4	1	L15
L14	l13 and l8	10	L14
L13	CD1$ adj blocking adj agent$	19	L13
L12	L11	1545875	L12
L11	cd1 adj blocking agent$1	1545875	L11
L10	l4 and l8L9	0	L10
L9	L8 and l3	1	L9
L8	L7 and (human$5)	526	L8
L7	anti-CD1$3 adj5 antibod$4	566	L7
L6	l3 and ((class adj switching) or (polyclonal adj3 activation))	0	L6
L5	L4 and l3	0	L5
L4	block adj cd1	9	L4
L3	treat adj (sle or lupus)	53	L3
L2	strober-samuel-$.in.	1	L2
L1	zeng-defu-$.in.	0	L1

END OF SEARCH HISTORY

(FILE 'HOME' ENTERED AT 15:45:41 ON 30 JAN 2003)

FILE 'EMBASE, BIOSIS, MEDLINE, CAPLUS, SCISEARCH' ENTERED AT 15:45:53 ON 30 JAN 2003

		E	ZENG DEFU?/AU
L1	32	S	E2
		E	STROBER SAMUEL?/AU
L2	117	S	E1 OR E2
L3	43	S	TREAT? (P) (SLE OR LUPUS) (P) ANTI-CD1?
L4	0	S	L3 AND (CLASS SWITCH?)
L5	16	DUP REM	L3 (27 DUPLICATES REMOVED)
L6	2	S	L5 AND BLOCKING
L7	1	S	L6 AND (HUMAN OR HUMANI?)
L8	1	S	(L1 OR L2) AND L3
L9	1	S	L8 NOT L7
L10	1	S	L8 NOT L6
L11	16	DUP REM	L5 (0 DUPLICATES REMOVED)
L12	1	S	L2 AND L3
L13	0	S	L3 AND (POLYCLONAL B CELL ACTIVATION)
L14	0	S	L3 AND (CD1 BLOCKING AGENT?)

L11 ANSWER 6 OF 16 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:258450 BIOSIS
DOCUMENT NUMBER: PREV200100258450
TITLE: CD1d1 deficiency does not affect in vivo anti-DNA antibody production and development of nephritis in MRL-lpr mice.
AUTHOR(S): Yang, Junqi (1); Liu, Hongzhu (1); van Kaer, Luc; Singh, Ram Raj (1)
CORPORATE SOURCE: (1) Autoimmunity and Tolerance Lab, Dept of Med, Univ. of Cincinnati and VAMC, Cincinnati, OH, 45220 USA
SOURCE: FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A1065. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Several lines of evidence suggest a role of CD1, a non-polymorphic antigen presenting molecule in autoantibody production and development of systemic **lupus** erythematosus (**SLE**): a) A NZB/W-derived T-NK cell line, when activated in the presence of CD1-transfected APCs, promotes IgG anti-DNA antibody production by syngeneic B cells in vitro (RR Singh, unpublished observation); b) TCR transgenic mice that have CD1-reactive T cells develop **lupus**-like syndrome (S Strober, 1997); c) **Anti-CD1** antibody **treatment** suppresses anti-DNA antibody production in **lupus**-prone NZB/W mice (D Zeng et al, 2000); and d) CD1c-reactive T cells from **SLE** patients promote IgG production in vitro (P Sieling et al, 2000). To confirm the role of CD1 in **lupus**, we backcrossed B6/129 CD1d1 mutant (CD1-/-) mice onto the MRL-1pr background for 9-10 generations. The final heterozygous mice were intercrossed to generate CD1-/- MRL-1pr mice. 16 CD1-/-, 20 CD1+/- and 14 CD1+/+ littermates were monitored for anti-DNA antibody production and proteinuria. Serum IgG anti-dsDNA antibody levels, proteinuria and renal histology were similar in the three groups of mice. These findings are at odds with previous reports that suggest a major role of CD1 in the pathogenesis of **SLE**. There are at least three possibilities to explain our findings: a) CD1 does not contribute to in vivo autoantibody production and **lupus** nephritis; b) CD1d2 molecule is involved in autoantibody production and development of **lupus**, or c) two populations of CD1-reactive T cells may co-exist in mice, one that promotes autoimmunity and the other that protects from autoimmunity. Investigations will analyze these possibilities.
AB. . . lines of evidence suggest a role of CD1, a non-polymorphic antigen presenting molecule in autoantibody production and development of systemic **lupus** erythematosus (**SLE**): a) A NZB/W-derived T-NK cell line, when activated in the presence of CD1-transfected APCs, promotes IgG anti-DNA antibody production by syngeneic B cells in vitro (RR Singh, unpublished observation); b) TCR transgenic mice that have CD1-reactive T cells develop **lupus**-like syndrome (S Strober, 1997); c) **Anti-CD1** antibody **treatment** suppresses anti-DNA antibody production in **lupus**-prone NZB/W mice (D Zeng et al, 2000); and d) CD1c-reactive T cells from **SLE** patients promote IgG production in vitro (P Sieling et al, 2000). To confirm the role of CD1 in **lupus**, we backcrossed B6/129 CD1d1 mutant (CD1-/-) mice onto the MRL-1pr background for 9-10 generations. The final heterozygous mice were intercrossed. . . mice. These findings are at odds with previous reports that suggest a major role of CD1 in the pathogenesis of **SLE**. There are at least three possibilities to explain our findings: a) CD1 does not contribute to in vivo autoantibody production and **lupus** nephritis; b) CD1d2 molecule is involved in autoantibody production and development of **lupus**, or c) two populations of CD1-reactive T cells may co-exist in mice, one that promotes autoimmunity and the other that. . .

L11 ANSWER 7 OF 16 SCISEARCH COPYRIGHT 2003 ISI (R)
ACCESSION NUMBER: 2002:105306 SCISEARCH
THE GENUINE ARTICLE: 498EB
TITLE: CD40-CD154 interactions in the pathogenesis of murine **lupus**: The beneficial effects of early and late **anti-CD154** antibody **treatment** appear to be mediated through different mechanisms.
AUTHOR: Burns C M (Reprint); Quesada S; Noelle R J; Schned A
SOURCE: ARTHRITIS AND RHEUMATISM, (SEP 2001) Vol. 44, No. 9, Supp. [S], pp. S397-+. MA 2062.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012 USA.
ISSN: 0004-3591.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0
TI CD40-CD154 interactions in the pathogenesis of murine **lupus**: The beneficial effects of early and late **anti-CD154** antibody **treatment** appear to be mediated through different mechanisms.

L11 ANSWER 8 OF 16 SCISEARCH COPYRIGHT 2003 ISI (R)
ACCESSION NUMBER: 2002:104621 SCISEARCH
THE GENUINE ARTICLE: 498EB
TITLE: Analysis of CD5 expression on peripheral B cells following **treatment** of active **SLE** patients with humanized **anti-CD154** mAb (5C8, BG9588).
AUTHOR: Gur H (Reprint); Lipsky P E; Shinohara S; Vazquez E; Illei G; Grammer A C
SOURCE: ARTHRITIS AND RHEUMATISM, (SEP 2001) Vol. 44, No. 9, Supp. [S], pp. S282-S282. MA 1376.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012 USA.
ISSN: 0004-3591.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0
TI Analysis of CD5 expression on peripheral B cells following **treatment** of active **SLE** patients with humanized **anti-CD154** mAb (5C8, BG9588).

L11 ANSWER 9 OF 16 SCISEARCH COPYRIGHT 2003 ISI (R)
ACCESSION NUMBER: 2002:104620 SCISEARCH
THE GENUINE ARTICLE: 498EB
TITLE: Normalization of peripheral B cells following **treatment** of active **SLE** patients with humanized **anti-CD154** mAb (5C8, BG9588).
AUTHOR: Grammer A C (Reprint); Shinohara N; Vazquez E; Gur H; Illei G; Lipsky P E
SOURCE: ARTHRITIS AND RHEUMATISM, (SEP 2001) Vol. 44, No. 9, Supp. [S], pp. S282-S282. MA 1375.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012 USA.
ISSN: 0004-3591.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0
TI Normalization of peripheral B cells following **treatment** of active **SLE** patients with humanized **anti-CD154** mAb (5C8, BG9588).

L11 ANSWER 10 OF 16 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001085839 EMBASE
TITLE: A humanized anti-human CD154 monoclonal antibody blocks CD154-CD40 mediated human B cell activation.
AUTHOR: Brams P.; Black A.; Padlan E.A.; Hariharan K.; Leonard J.; Chambers-Slater K.; Noelle R.J.; Newman R.
CORPORATE SOURCE: R. Newman, IDEC Pharmaceuticals Corporation, 3010 Science Park Road, San Diego, CA 92121, United States. rnewman@idecpharm.com
SOURCE: International Immunopharmacology, (2001) 1/2 (277-294). Refs: 46
ISSN: 1567-5769 CODEN: IINMBA
PUBLISHER IDENT.: S 1567-5769(00)00020-5
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Humanized **anti-CD154** antibody, IDEC-131, had a slightly, but reproducibly, better binding affinity for CD154 (K(d)= 5.6 nM), compared to the parent antibody 24-31 (K(d)= 8.5 nM). Otherwise it was indistinguishable from the murine parent antibody in its ability to bind to CD154, block CD154 binding to CD40 and inhibit T cell-dependent B cell differentiation. The latter activity was independent of FcR binding as the Fab'1 fragment of IDEC-131 had an equivalent biological activity to that of the whole antibody. IDEC-131 blocked soluble CD 154 from inducing proliferation of purified B cells, and blocked T cell dependent anti-tetanus toxoid specific antibody production by human B cells in vitro. IDEC-131, .gamma.1, .kappa., had strong Fc.gamma.RI, Fc.gamma.RII and C1q binding, but was unable to induce complement dependent (CDC) or antibody dependent cell-cytotoxicity (ADCC) of activated peripheral blood T cells, which express relatively low levels of CD154. IDEC-131 antibody inhibited both primary and secondary antibody responses to ovalbumin in cynomolgus monkeys at a dose of 5 mg/kg. In non-immunized animals, **treatment** with IDEC-131 at 50 mg/kg weekly for 13 weeks induced no change in any of the measured lymphocyte subsets, including B cells, CD4 + and CD8 + T cells. Similarly, a safety study in chimpanzees showed no discernible safety related issues at 20 mg/kg, including B and T cell subsets. These results show that the humanized **anti-CD154** antibody, IDEC-131, has retained the affinity and functional activity of its routine parent antibody, is unlikely to deplete CD154 positive lymphocytes in humans, and is safe and effective in blocking antibody production in monkeys. Based on its safety and efficacy profile, IDEC-131 is being developed for therapy of systemic **lupus** erythematosus. .COPYRGT. 2001 Elsevier Science B.V.
AB Humanized **anti-CD154** antibody, IDEC-131, had a slightly, but reproducibly, better binding affinity for CD154 (K(d)= 5.6 nM), compared to the parent antibody. . . both primary and secondary antibody responses to ovalbumin in cynomolgus monkeys at a dose of 5 mg/kg. In non-immunized animals, **treatment** with IDEC-131 at 50 mg/kg weekly for 13 weeks induced no change in any of the measured lymphocyte subsets, including. . . no discernible safety related issues at 20 mg/kg, including B and T cell subsets. These results show that the humanized **anti-CD154** antibody, IDEC-131, has retained the affinity and functional activity of its routine parent antibody, is unlikely to deplete CD154 positive. . . blocking antibody production in monkeys. Based on its safety and efficacy profile, IDEC-131 is being developed for therapy of systemic **lupus** erythematosus. .COPYRGT. 2001 Elsevier Science B.V.

L11 ANSWER 11 OF 16 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000176484 EMBASE
TITLE: Cutting edge: A role for CD1 in the pathogenesis of lupus

in NZB/NZW mice.

AUTHOR: Zeng D.; Lee M.; Tung J.; Brendolan A.; Strober S.
CORPORATE SOURCE: Dr. S. Strober, Div. of Immunology and Rheumatology, Stanford Univ. School of Medicine, 300 Pasteur Drive, Stanford, CA 94305, United States. sstrober@stanford.edu

SOURCE: Journal of Immunology, (15 May 2000) 164/10 (5000-5004).
Refs: 28
ISSN: 0022-1767 CODEN: JOIMA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
031 Arthritis and Rheumatism
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Since **anti-CD1** TCR transgenic T cells can activate syngeneic B cells via CD1 to secrete IgM and IgG and induce **lupus** in BALB/c mice, we studied the role of CD1 in the pathogenesis of **lupus** in NZB/NZW mice. Approximately 20% of B cells from the spleens of NZB/NZW mice expressed high levels of CD1 (CD1(high) B cells). The latter subset spontaneously produced large amounts of IgM anti-dsDNA Abs in vitro that was up to 25-fold higher than that of residual CD1(int/low) B cells. T cells in the NZB/NZW spleen proliferated vigorously to the CD1-transfected A20 B cell line, but not to the parent line. **Treatment** of NZB/NZW mice with **anti-CD1** mAbs ameliorated the development of **lupus**. These results suggest that the CD1(high) B cells and their progeny are a major source of autoantibody production, and activation of B cells via CD1 may play an important role in the pathogenesis of **lupus**.

AB Since **anti-CD1** TCR transgenic T cells can activate syngeneic B cells via CD1 to secrete IgM and IgG and induce **lupus** in BALB/c mice, we studied the role of CD1 in the pathogenesis of **lupus** in NZB/NZW mice. Approximately 20% of B cells from the spleens of NZB/NZW mice expressed high levels of CD1 (CD1(high). . . cells in the NZB/NZW spleen proliferated vigorously to the CD1-transfected A20 B cell line, but not to the parent line. **Treatment** of NZB/NZW mice with **anti-CD1** mAbs ameliorated the development of **lupus**. These results suggest that the CD1(high) B cells and their progeny are a major source of autoantibody production, and activation of B cells via CD1 may play an important role in the pathogenesis of **lupus**.

=>

L8 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:325679 CAPLUS
DOCUMENT NUMBER: 133:72828
TITLE: Cutting edge: a role for CD1 in the pathogenesis of lupus in NZB/NZW mice
AUTHOR(S): **Zeng, Defu**; Lee, Mi-Kyeong; Tung, James; Brendolan, Andrea; **Strober, Samuel**
CORPORATE SOURCE: Department of Medicine, Division of Immunology and Rheumatology, Stanford University School of Medicine, Stanford, CA, 94306, USA
SOURCE: Journal of Immunology (2000), 164(10), 5000-5004
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

May issue

X

AB Since **anti-CD1** TCR transgenic T cells can activate syngeneic B cells via CD1 to secrete IgM and IgG and induce **lupus** in BALB/c mice, we studied the role of CD1 in the pathogenesis of **lupus** in NZB/NZW mice. Approx. 20% of B cells from the spleens of NZB/NZW mice expressed high levels of CD1 (CD1high B cells). The latter subset spontaneously produced large amts. of IgM anti-dsDNA Abs in vitro that was up to 25-fold higher than that of residual CD1int/low B cells. T cells in the NZB/NZW spleen proliferated vigorously to the CD1-transfected A20 B cell line, but not to the parent line. **Treatment** of NZB/NZW mice with **anti-CD1** mAbs ameliorated the development of **lupus**. These results suggest that the CD1high B cells and their progeny are a major source of auto-antibody prodn., and activation of B cells via CD1 may play an important role in the pathogenesis of **lupus**.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AU **Zeng, Defu**; Lee, Mi-Kyeong; Tung, James; Brendolan, Andrea; **Strober, Samuel**

AB Since **anti-CD1** TCR transgenic T cells can activate syngeneic B cells via CD1 to secrete IgM and IgG and induce **lupus** in BALB/c mice, we studied the role of CD1 in the pathogenesis of **lupus** in NZB/NZW mice. Approx. 20% of B cells from the spleens of NZB/NZW mice expressed high levels of CD1 (CD1high B cells). The latter subset spontaneously produced large amts. of IgM anti-dsDNA Abs in vitro that was up to 25-fold higher than that of residual CD1int/low B cells. T cells in the NZB/NZW spleen proliferated vigorously to the CD1-transfected A20 B cell line, but not to the parent line. **Treatment** of NZB/NZW mice with **anti-CD1** mAbs ameliorated the development of **lupus**. These results suggest that the CD1high B cells and their progeny are a major source of auto-antibody prodn., and activation of B cells via CD1 may play an important role in the pathogenesis of **lupus**.

=>

PharMingen

Anti-Mouse CD1d, Clone 1B1

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DESCRIPTION

The 1B1 antibody reacts with mouse CD1d, a 48-kDa glycoprotein with structural homology to the major histocompatibility complex (MHC) class I molecules.1 Like MHC class I, CD1d associates with beta2-microglobulin, but similar to MHC class II, CD1d binds preferentially to long peptides with hydrophobic residues at specific positions.2 The mouse Cd1 gene family consists of two highly similar genes Cd1.1 and Cd1.2, which are homologous to human Cd1d.3 CD1d+ cortical thymocytes are postulated to mediate the positive selection of NK1+ T cells, which have a limited T-cell receptor repertoire and the exceptional ability to produce cytokines in response to primary stimulation.4,5 CD1d expression has been detected by mAb 1B1 at varying levels on most types of bone marrow and peripheral leukocytes and on epithelial, dendritic, and lymphoid cells in the thymus.1,6 The 1B1 antibody competes with mAb 1H1 (another antibody specific for mouse CD1d7) in binding to mouse splenocytes.6

USAGE

This antibody has been tested by immunofluorescent staining (lesser than or greater to 4 µg/ million cells) with flow cytometric analysis to assure specificity and reactivity. Other reported applications include immunohistochemical staining of acetone-fixed frozen sections.6 Since applications vary, each investigator must determine dilutions appropriate for individual use.

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REFERENCES

1. Sydora, B.C., L. Brossay, A. Hagenbaugh, M. Kronenberg, and H. Cheroutre. 1996. TAP-independent selection of CD8+ intestinal intraepithelial lymphocytes. *J. Immunol.* 156: 4209 - 4216.
2. Casta–o, A.R., S. Tangri, J.E.W. Miller, H.R. Holcombe, M.R. Jackson, W.D. Huse, M. Kronenberg, and P.A. Peterson. 1995. Peptide binding and presentation by mouse CD1. *Science* 269: 223 - 226.
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7. Balk, S.P., S. Burke, J.E. Polischuk, M.E. Frantz, L. Yang, S. Porcelli, S.P. Colgan, and R.S. Blumberg. 1994. b2-microglobulin-independent MHC Class Ib molecule expressed by human intestinal

epithelium. *Science* 265: 259 - 262.

Cat. No.	Description	Clone	Size
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09902D	Biotinylated anti-mouse CD1d	1B1	0.5 mg
09904D	FITC labeled anti-mouse CD1d	1B1	0.5 mg
09905A	PE labeled anti-mouse CD1d	1B1	0.2 mg

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- **NK cells (PK136)**
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- **TER-119**

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